Psychogenic Lowering of Urinary Cortisol Levels Linked to Increased Emotional Numbing and a Shame-Depressive Syndrome in Combat-Related Posttraumatic Stress Disorder

JOHN W. MASON, MD, SHEILA WANG, PHD, RACHEL YEHUDA, PHD, SHERRY RINEY, MSW, DENNIS S. CHARNEY, MD, AND STEVEN M. SOUTHWICK, MD

Objective: The purpose of the study was to search for the intrapsychic correlates of individual differences in cortisol levels in male Vietnam combat veterans with posttraumatic stress disorder. **Methods:** The study involved measurement of urinary cortisol levels and clinical assessment with a broad profile of psychometric tests during a single 48-hour period in 30 inpatients. **Results:** The main finding by both correlation and *t* test analyses was a significant inverse relationship between urinary cortisol levels and a symptom complex composed of two closely interrelated clinical subgroupings, "disengagement" (principally involving emotional numbing) and "shame-laden depression." **Conclusions:** The findings support the concept that cortisol levels reflect the ongoing balance between the undifferentiated emotional arousal state of engagement (associated with higher cortisol levels) and opposing antiarousal disengagement defense mechanisms (associated with lower cortisol levels). It appears that the low cortisol levels often seen in patients with posttraumatic stress disorder are psychogenic and reflect a dominating effect of disengagement coping strategies, which represent secondary compensatory adaptations during the chronic course of this disorder to counteract primary arousal symptoms, especially those related to an intractable shame-laden depressive syndrome. The psychoendocrine findings suggest that the relatively inconspicuous clinical feature of shame resulting from both the primary and secondary traumatizations is a particularly powerful, preoccupying, and overwhelming source of emotional engagement. Shame may represent a "sleeper" that is worthy of greater attention in both research and clinical efforts to understand the pathogenesis and psychopathology of this devastating stress-related disorder. **Key words:** posttraumatic stress disorder, cortisol, emotional numbing, shame, guilt, depression.

BDS = Beck Depression Scale; BPRS = Brief Psychiatric Rating Scale; CAPS-2 = Clinician-Administered PTSD Scale; CES = Combat Exposure Scale; DEQ = Depressive Experiences Questionnaire; HDS = Hamilton Depression Scale; MMPI = Minnesota Multiphasic Personality Inventory; PTSD = posttraumatic stress disorder; VA = Veterans Affairs.

INTRODUCTION

An early pilot psychoendocrine survey of male Vietnam combat veterans with PTSD, studied in the routine general psychiatry service setting of the West Haven VA Medical Center, revealed the unexpected finding of a relatively low mean urinary free cortisol level (33 μg/d) in comparison with several other diagnostic groups of psychiatric patients, including those with major depressive disorder, bipolar manic disorder, and undifferentiated schizophrenia (1). This finding was somewhat puzzling, not only because of the common occurrence of elevation of cortisol levels in relation to stress but also because the low levels of cortisol in these PTSD patients were observed paradoxically in the face of elevated urinary norepinephrine and epinephrine levels. In other words, there was a striking dissociation in these PTSD patients between the pituitary-adrenal-cortical and the sympathetic-adrenal-medullary systems, both of which typically show concurrently increased activity in relation to stress (2–4). In a follow-up study at the same VA medical center, mean urinary cortisol levels were again found to be relatively low in two additional samples of veterans with combat-related PTSD, 43.3 μg/d in a sample of nine inpatients and 37.7 μg/d in a sample of seven outpatients, both significantly lower than the 62 μg/d value of a comparison group of normal control subjects (5).

Most subsequent work in the field that followed this original discovery has been approached from a largely biological perspective, especially with challenge test approaches looking for biological abnormalities in hypothalamic-pituitary-adrenal axis function and biological diagnostic markers for PTSD. The conceptual approach underlying our clinical psychoendocrine studies, however, places great emphasis on the importance of including consideration of psychosocial factors and a potential psychogenic basis for cortisol alterations in stress-related disorders. A massive body of

From the Department of Psychiatry, Yale University School of Medicine, New Haven, and the National Center for PTSD, Clinical Neuroscience Division, Veterans Affairs Medical Center (J.W.M., S.W., D.S.C., S.M.S.), West Haven, Connecticut; the Department of Psychiatry, Mount Sinai Medical School, New York, and Bronx Veterans Affairs Hospital (R.Y.), Bronx, NY; and the National Center for PTSD, Clinical and Educational Division, Menlo Park Veterans Affairs Medical Center (S.R.), Palo Alto, California.

Address reprint requests to: John W. Mason, MD, 32 Maple Vale Dr., Woodbridge, CT 06525. Email: jwmason@pol.net

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evidence in early stress research has established that cortisol levels are exquisitely sensitive to psychosocial influences, even very subtle intrapsychic and social influences, and that psychosocial factors are the most potent and prevalent stressful influences on the cortisol system in everyday life (6–9).

It was in this historical perspective that the findings of the pilot PTSD study forcefully brought to mind many early basic stress research studies in both humans and animals. Those studies revealed in a similar way surprisingly low cortisol levels in stressful situations where one might have expected cortisol levels to be quite high. In 1957, in one of the earliest clinical psychoendocrine studies, it was found that most patients anticipating elective cardiac surgery had relatively low preoperative cortisol levels rather than the marked elevations that might have been expected in such a gravely threatening situation, and it was observed that these patients were those who were able to use "disengaging" coping strategies such as avoidance, withdrawal, or denial in the face of the impending surgery, which had a high mortality rate (10). On the other hand, those preoperative patients who were able to become "engaged" or to "make active emotional participation" with the serious reality of their stressful experience showed increased cortisol levels. From this study, which used both Rorschach testing and clinical observation, Margaret Thaler Singer developed the new construct of engagement, or engagement-involvement, as a relatively broad and undifferentiated arousal state (11) and introduced the concept of an engagement-nonengagement axis as the primary underlying intrapsychic dimension that seemed to be directly reflected in the cortisol system.

Another closely related early psychoendocrine study leading to the same conclusions was that involving the parents of leukemic children, in which surprisingly low corticosteroid levels were observed in a subgroup of parents during the course of the fatal illness of their child. In such "low mean cortisol parents," it was found that cortisol levels tended to go still lower when acute stress was superimposed on the chronic baseline stress, suggesting the operation of an active suppressive psychogenic mechanism as the basis for the low cortisol levels. Further psychiatric observations revealed that such low-cortisol parents were those who typically used disengagement coping strategies, particularly denial and avoidance, often in rather flagrant ways. With this lead, the psychiatric team, using a semistructured interview with good interobserver reliability, was able in a prospective study to predict successfully the cortisol levels of individual parents on the basis of assessing the effectiveness of psychological defenses, again supporting the concept of the balance of opposing forces in the engagement-disengagement axis as the primary underlying intrapsychic dimension linked to the cortisol system (12, 13).

Another related early example, in this case involving a direct military setting, was that of a Special Forces "A" Team in Vietnam. This longitudinal study showed that the men who used disengagement coping strategies had lower cortisol levels on the day they expected a massive overrunning attack on their outpost by the Vietcong than on the days before and after the expected attack, whereas the men who were forced to remain engaged with the life-threatening situation because of the nature of their duties (the officer and the radio operator) showed elevated cortisol levels (14), again supporting the validity of Singer's formulation implicating the prominent role of the engagement-disengagement axis in cortisol regulation.

These and many other subsequent basic studies in both animals and humans have made it extremely clear that cortisol levels reflect not only emotional arousal but also active defensive or antiarousal intrapsychic mechanisms (4, 9) and should be conceptualized in a psychosocial perspective as representing at any given point in time a balance between the opposing intrapsychic forces of the engagement-disengagement axis. It should also be emphasized that strong support for the psychogenic basis underlying the lowering of cortisol levels observed in the early stress studies described above was provided by longitudinal data showing a close time relationship between occurrence of acute superimposed psychosocial stress and coincidental lowering of cortisol levels.

Therefore, as we proceeded to plan follow-up research based on our pilot study findings, an early objective was to determine whether the psychogenic influence of disengagement coping strategies might be associated with the low cortisol levels in PTSD and thereby illuminate the clinical meaning of this hormonal alteration. As resources at the National Center for PTSD became available, the large scale, complexity, and intensity of the multiproject research program in the West Haven Division precluded rigorous, custom-designed correlational psychoendocrine studies. But an opportunity eventually did develop to set up the present dedicated study at the Menlo Park Division in a way that promoted more incisive conditions for correlational investigation, especially in being able to obtain hormonal and psychometric measurements as close to the same point in time as possible (within the same 48-hour calendar period) and in the same social setting for all patients. In keeping with the historical rationale outlined above, the main purpose of the present study was to scrutinize correlational data for relationships between the levels of cortisol and clini-

cal psychological variables that might reflect either engagement or disengagement mechanisms, which are known from early basic psychoendocrine research to be closely linked to the cortisol system.

METHODS

The sample was composed of 30 male Vietnam combat veterans with PTSD who were inpatients in an elective treatment program at the Menlo Park Division of the National Center for PTSD. The diagnosis was established using the Structured Clinical Interview for DSM-III-R (15). Exclusion criteria included major medical illnesses, hormonal medication, psychotic disorders, organic brain syndrome, and current drug or alcohol abuse less than 3 months before the study. After obtaining informed consent, hormonal and psychometric assessments were made during the same 2-day period on all patients. Because of the large number of patients being studied simultaneously during a 2-day period, clinician-administered psychometric testing was done by a team of experienced clinicians who were trained in advance for the purpose of promoting consistency and standardization in the assessments.

During the 2-day period of 24-hour urine collections, core PTSD symptoms were measured using the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (16) and CAPS-2 (17). Additional measurements included the BPRS (18), the HDS (19), the BDS (20), and the DEQ (21, 22). Measurements available to us from the hospital admission period included the CES (23) and the MMPI-2, including the Clinical, Harris-Lingoes, Content, and Supplementary Scales (24).

Mean values ± SEM that characterize this patient sample from a demographic and clinical standpoint were as follows: age = 42.4 ± 0.9 years, weight = 197 ± 6 lb, height = 70.8 ± 0.5 inches, CES score = 26.0 ± 1.5, Mississippi total score = 122.7 ± 3.2, Mississippi Keane emotional numbing factor (16) = 26.1 ± 0.7, Mississippi King numbing-withdrawal factor (25) = 41.3 ± 1.0, CAPS-2 intensity sum = 32.7 ± 1.4, CAPS-2 reexperiencing intensity subscore = 6.8 ± 0.6, CAPS-2 avoidance intensity subscore = 13.7 ± 0.7, CAPS-2 hyperarousal intensity subscore = 12.2 ± 0.7, BPRS sum = 17.6 ± 1.2, BPRS guilt feelings = 2.53 ± 0.3, HDS (46 items) = 27.2 ± 1.8, HDS (21 items) = 17.4 ± 1.2, BDS = 21.5 ± 1.5, DEQ self-criticism factor = 1.14 ± 0.1, and MMPI-2 Content Scale depression score = 86.0 ± 2.1. The sample was 46% white, 42% Hispanic, and 8% black, but no significant differences were observed in cortisol levels between the racial subgroups. With regard to comorbidity, diagnostic criteria were met by 59% for prior alcohol abuse, 70% for recurrent major depressive disorder, and 19% for panic disorder. Because of a practical need to obtain patients whose level of social and clinical functioning was sufficiently high for them to tolerate and participate in an intensive, long-term, group-oriented clinical and research program, this global clinical selection bias tended to reduce the likelihood of including patients with frequent and severe socially disabling dissociative symptoms, such as dissociative fugue states or identity disorders, or marked borderline personality disorder.

Two consecutive 24-hour urine samples were collected in 3-liter amber polypropylene containers. The containers were kept in a freezer at −20°C throughout the collection periods to minimize loss of hormone through deterioration. After standardized, rapid thawing and shaking of the frozen 24-hour urine samples, 2-ml untreated urine aliquots of each sample were saved for the cortisol assays and frozen. These frozen aliquots were immediately shipped in dry ice to the West Haven Division of the National Center for PTSD and were then kept frozen at −70°C until the hormonal assays were performed about 1 week later. Free cortisol excretion rate was measured using a radioimmunoassay kit from Incstar Corporation (now Diasporin Corp., Stillwater, MN); an interassay coefficient of variation of 4.0% was obtained in our laboratory.

The mean ± SEM urinary free cortisol level was 61.3 ± 3.9 μg/d on the first day and 60.5 ± 3.2 μg/d on the second day. There was no significant difference between values obtained on the 2 days (t = 0.24, $p < .8$), so the mean of these two samples was used in all correlational analyses. Two patients with extremely high outlying cortisol values (>120 μg/d, 25 μg/d above the next highest patient) were eliminated from the sample, as were three patients with extremely low outlying CAPS-2 sum values. Although inclusion of these patients would not change the significance of the main findings or the conclusions derived from this study, the refined sample seems to present a more representative picture of the relationships between hormonal and clinical variables in this patient group.

RESULTS

To protect against the vagaries of correlational analyses and the risk of spurious findings when dealing with isolated individual items or factors in a single psychometric instrument, we included multiple diverse, overlapping scales in our test battery, which used clinician-administered as well as self-reported procedures and both state and characterological measures. A main purpose of this approach was to determine whether a finding with any single score could be confirmed by a larger weight of evidence with similar items or factors as assessed independently by other psychometric instruments.

When Pearson product-moment correlation coefficients were determined for the association between mean urinary cortisol levels and the overall battery of psychometric test scores, a remarkably large number of significant correlations were found, far exceeding those observed in relation to any other hormonal or biological measures in our study. In an initial screening of five psychometric instruments providing a total matrix of the 116 clinical variables used in this study, there were a total of 25 significant ($p < .05$) correlations with urinary cortisol levels, whereas with 10 other hormonal measures the total number of significant correlations ranged from only 1 to 6 for the different hormones (mean of 3) or only about 14% of the significant correlations shown by cortisol. It seems likely, therefore, that the large number of significant correlations involving cortisol levels cannot be dismissed or accounted for simply on the basis of the total number of clinical variables being compared with the cortisol values, because all of the other 10 hormonal measures (showing far fewer significant correlations) were compared with exactly the same total number of clinical measures as cortisol.

Another striking feature of the significant cortisol correlations is that at the initial inspection stage of analysis virtually all of them were found to be consis-

tently inverse and to fall remarkably into three clinically distinct subgroupings: 1) disengagement-emotional numbing, 2) self-critical depression, and 3) shame-guilt. This very specific, orderly, and clinically meaningful organization of cortisol-psychometric correlates into only three homogeneous clinical categories, along with the uniformly inverse direction of the correlations, also seems to militate convincingly against the possibility of random significance of the cortisol correlations simply as a function of the number of clinical variables involved in the correlational analyses. Furthermore, the clinical rationale for conceptualizing psychopathological interrelationships of these three subgroupings as logical parts of a unified clinical or symptom complex is very compelling and is discussed later.

Correlations Between Cortisol and Core PTSD Symptoms

The data analysis was focused first on the core symptoms of PTSD as measured by the CAPS-2 scale because this test provides a major factor subscore for avoidance-numbing symptoms, which represent disengagement coping mechanisms of the type believed to be associated with low cortisol levels from prior work in the psychoendocrine field. CAPS-2 is a clinician-administered test composed of 30 items, the first 17 of which are directed at core symptom assessment and are divided into three (B, C, and D) categorical factors or subscores.

Table 1 shows the correlations between cortisol levels and these major subscores for the intensity of PTSD core symptoms as well as the total score for all 17 items. Of the three core symptom categories, only C (avoidance-numbing) shows a significant correlation with urinary cortisol levels ($r = -0.381$, $p < .04$). It is evident that the significant correlation ($r = -0.385$, $p < .04$) between the total PTSD symptom score and cortisol levels largely reflects the significant correlation between cortisol levels and the avoidance-numbing subscore because the reexperiencing ($p < .3$) and hyperarousal ($p < .2$) subscores are not close to significance. Note that the significant correlations are inverse, indicating that the lower the level of cortisol, the higher the level of avoidance-numbing, just as would have been predicted by the Singer formulation reviewed above. Further inspection of the correlation results indicates that CAPS-2 item 10, restricted range of affect, a measure of emotional numbing showing a highly significant correlation ($r = -0.520$, $p < .003$) with cortisol, largely accounts for the significant C subscore correlation. Thus, this CAPS-2 finding may be regarded as primarily representing an emotional numbing relationship with cortisol levels in this patient sample.

TABLE 1. Correlations Between Urinary Cortisol Levels and the Intensity of CAPS-2 PTSD Core Symptom Subscores

CAPS-2 Subscore	*r*	*p*
B (reexperiencing)	−0.257	<.26
C (avoidance-numbing)	−0.381	<.04
D (hyperarousal)	−0.219	<.24
Total PTSD symptoms score	−0.385	<.04

Additional Correlations Between Cortisol and Numbing and Disengagement

On the basis of the above CAPS-2 finding of a significant inverse correlation between cortisol levels and a measure of avoidance-numbing, reflecting disengagement coping strategies in this PTSD patient sample, we next explored other similar measurements available from our test battery to determine whether this finding might receive further support across different psychometric scales. Table 2 summarizes this survey, showing that a substantial number of standard items or factors from the CAPS-2, Mississippi, and BPRS scales reflecting disengagement defenses correlate with cortisol levels at or near a statistically significant level.

As mentioned above, the strongest correlation is that with the CAPS-2 restricted range of affect measure ($r = -0.520$, $p < .003$). In addition, the BPRS blunted affect item showed a significant correlation with cortisol levels ($r = -0.384$, $p < .04$). The strongest correlation with any single Mississippi item ($r = -0.472$, $p < .01$) is with item 22 ("I do not enjoy the company of others"). All of these items from the three different scales reflect a disposition toward intrapsychic or social disengagement.

With regard to broader "factor" scores, which generally provide greater weight than individual item scores, it was found that although the Keane Mississippi emotional numbing factor did not quite reach significance ($r = -0.358$, $p < .06$), the King Mississippi numbing-withdrawal factor did correlate significantly with cortisol levels ($r = -0.422$, $p < .02$); in fact, the latter correlation was the second strongest correlation of any single Mississippi item or factor score.

Other factors in this overall subgrouping that showed significant correlations include the previously mentioned CAPS-2 avoidance-numbing (C) subscore ($r = -0.381$, $p < .04$) and the BPRS withdrawal-retardation factor ($r = -0.379$, $p < .04$).

Within this general disengagement category, which included emotional numbing, avoidance, and with-

TABLE 2. Correlations Between Urinary Cortisol Levels and Psychometric Measures of Disengagement Coping Strategies (Emotional Numbing, Avoidance, and Withdrawal)

Disengagement Subgrouping	*r*	*p*
Standard psychometric measures		
CAPS-2 avoidance-numbing factor	−0.381	<.038
Mississippi King numbing-withdrawal factor	−0.422	<.023
BPRS withdrawal-retardation factor	−0.379	<.039
CAPS-2 item 10 (restricted affect)	−0.527	<.003
Mississippi item 22 ("I don't enjoy the company of others")	−0.472	<.010
BPRS item 16 (blunted affect)	−0.384	<.036
Mississippi item 6 ("I can't be emotionally close with others")	−0.365	<.051
Mississippi Keane emotional numbing factor	−0.358	<.057
Improvised psychometric factors		
Multiscale disengagement factor[a]	−0.535	<.002
Multiscale disengagement factor 2[b]	−0.600	<.0005
Mississippi disengagement factor[c]	−0.398	<.032

[a] Sum of CAPS-2 avoidance-numbing factor, Mississippi King numbing-withdrawal factor, and BPRS withdrawal-retardation factor.
[b] Sum of CAPS-2 item 10 score, Mississippi King numbing-withdrawal factor, and BPRS withdrawal factor.
[c] Sum of items 1, 3, 5, 6, 9, 15, 16, 22, 23, 26, 28, 29, 30, and 35 from the Mississippi scale.

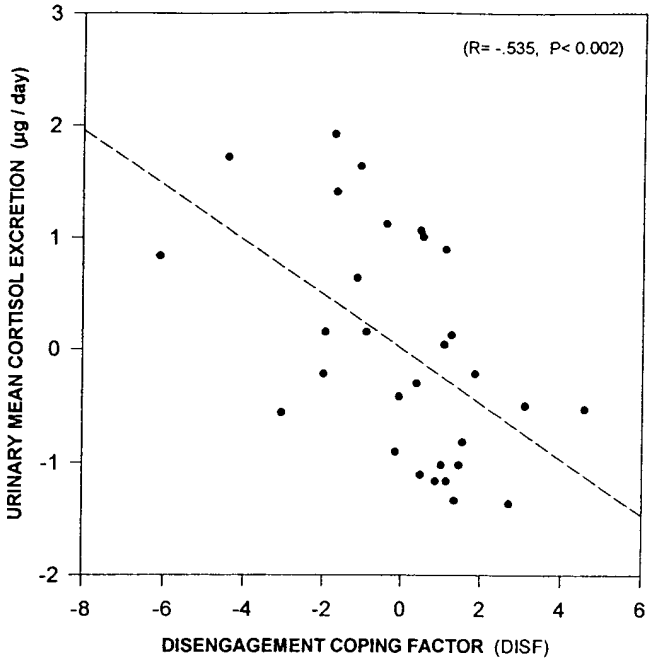

Fig. 1. Correlation between urinary cortisol levels and the use of emotional numbing-disengagement coping strategies in male Vietnam combat veterans with PTSD. DISF = sum of factors from CAPS-2, Mississippi, and BPRS scales (including emotional numbing, avoidance, and withdrawal measures).

drawal, it seems from the *p* values that those items reflecting emotional numbing, like the CAPS-2 restricted affect, the BPRS blunted affect, and the King numbing-withdrawal factor from the Mississippi scale, are most prominently and significantly correlated with cortisol levels.

Because none of these individual standard instruments provided designated factors for the broader measurement of disengagement as a more comprehensive coping category according to Singer's concept, we explored two possible ways of further testing or strengthening this finding by improvising some factors for a more inclusive assessment of this coping category in our patient sample. The first approach was simply to create a disengagement factor by using the sum of the scores of the factor correlates from three different psychometric scales (CAPS-2 avoidance-numbing factor, Mississippi King numbing-withdrawal factor, and BPRS withdrawal-retardation factor), shown in Table 2, to combine the breadth, weight, and cross-confirmatory power of the multiple cortisol correlates. When this multiscale "disengagement factor" was placed in a standardized score matrix with cortisol values, the correlation scatterplot, as shown in Figure 1, reveals a very high level of significance ($r = -0.535$, $p < .002$) and shows no extreme outliers accounting for the significance level.

As a second exploratory approach, the significant correlations of cortisol levels with Mississippi items 22 ("I do not enjoy company of others") and 6 ("I can't be emotionally close with others") suggested the possible usefulness of creating a "Mississippi disengagement factor" using 14 similar items selected from the Mississippi scale (items 1, 3, 5, 6, 9, 15, 16, 22, 23, 26, 28, 29, 30, and 35). The selected items are regarded from a clinical standpoint as reflecting a typical or dispositional coping inclination toward disengagement characterized by feelings of not being like other people; feeling alienated, mistrustful, and socially uncomfortable; and using emotional numbing, avoidance, or withdrawal coping strategies.

This improvised factor (Mississippi disengagement factor) also correlated significantly with urinary cortisol levels ($r = -0.398$, $p < .03$), but it did not improve on the King numbing-withdrawal factor correlation ($p < .02$), although it did yield a more significant correlation than did the Keane emotional numbing factor ($p < .06$). When the scores of the 21 Mississippi items not included in the improvised Mississippi disengagement factor were combined, the resulting factor did not show a significant correlation with cortisol levels ($r = -0.290$, $p < .13$), indicating that the finding was not simply a nonspecific reflection of the overall severity of symptoms picked up in the 14 items used.

Correlations Between Cortisol Levels and Self-Critical Depression

Because emotional numbing has been considered by some clinicians to represent a secondary clinical adaptive complex brought in to counteract a primary complex of severely distressing intrusive symptoms, we examined next our correlational matrix to see if the data might provide leads to the primary complex symptoms linked to emotional numbing and cortisol in this sample of patients. Remarkably, virtually all the remaining psychometric scores showing significant correlations with cortisol beyond the disengagement-emotional numbing category fell very naturally into two clinical subgroupings, a "self-critical depressive syndrome" and a "shame-guilt" category.

Table 3 summarizes the correlational findings between urinary cortisol levels and an array of different psychometric measurements of depressive symptoms, including several widely used depression scales. Of all the diverse depression scales studied, the depression score that showed the most significant correlation with cortisol levels was one that is relatively little used in psychiatric research, namely the MMPI Content Depression Scale ($r = -0.409$, $p < .03$). The negative correlation, of course, indicates that the greater the degree of depressive tendencies, the lower the cortisol level, quite in contrast to the general findings in many studies of psychiatric patients with major depressive disorder showing elevated cortisol levels.

At the same time, in this sample of patients with combat-related PTSD, the most widely used depression scales, including the HDS (both 21- and 46-item scoring), the BDS, and the standard MMPI-2 Clinical Depression Scale, did not show significant correlations with cortisol levels, although it is noteworthy that they all show negative rather than positive r values.

This interesting finding seems to indicate that the MMPI-2 Content Depression Scale is detecting a depressive syndrome or subtype that is qualitatively different from the clinical syndrome or syndromes measured by the more commonly used depression scales. In support of this view, as shown in Table 3, it is evident that the self-criticism factor of the DEQ also correlates significantly with cortisol levels ($r = -0.374$, $p < .04$). This finding seems to fit very well with the fact that the MMPI-2 Content Depression Scale is especially heavily loaded with items reflecting a deep sense of guilt and shame as manifested in self-deprecatory or self-condemning thoughts, such as "I believe my sins are unpardonable," "I believe I am a condemned person," "I do many things which I regret afterward," "At times I think I am no good at all," and "I have not lived the right kind of life."

To learn more about the meaning of this MMPI-2 Content Depression Scale finding, an analysis was done of the intercorrelations between this score and other clinical psychometric measures in our matrix. Table 4 shows the substantial number of very strong correlates discovered (these correlates are largely characterological measures from the MMPI-2 Content De-

TABLE 3. Correlations Between Urinary Cortisol Levels and Measures of Depression From Different Psychometric Scales

Depression Subgrouping	r	p
Standard psychometric measures		
MCD	−0.409	<.025
DEQ self-criticism score	−0.374	<.042
Mississippi item 15 (I feel like I can't go on")	−0.416	<.025
MMPI-2 Clinical Scale Depression Score	−0.308	<.097
Hamilton Depression Scale (46-item total score)	−0.239	<.204
Beck Depression Scale score	−0.218	<.247
Hamilton Depression Scale (21-item total score)	−0.128	<.500
Improvised psychometric factors		
Multiscale self-critical depression factor[a]	−0.497	<.005
CAPS-2 MCD-derived depression factor[b]	−0.405	<.026
Mississippi MCD-derived depression factor[c]	−0.375	<.045

MCD = MMPI-2 Content Depression Scale score.
[a] Sum of the MCD score, DEQ self-criticism score, and score on Mississippi item 15.
[b] Sum of CAPS-2 items correlating closely with MCD score (items 9, 10, 11, 13, 16, 17, 23, 24, 27, and 29).
[c] Sum of Mississippi items correlating closely with MCD score (items 3, 5, 8, 12, 13, 15, 16, 18, 26, 28, 34, and 35).

TABLE 4. Psychometric Correlates of the MMPI-2 Content Scale Depression Score in Vietnam Combat Veterans With PTSD

	r	p
MMPI-2 characterological measures		
Social introversion	0.669	<.0001
Social discomfort	0.614	<.0003
Low self-esteem	0.586	<.0007
Paranoia	0.695	<.0001
Anger	0.630	<.0002
Family problems	0.570	<.001
Work interference	0.813	<.0001
College maladjustment	0.831	<.0001
Other shame-guilt measures		
DEQ self-criticism score	0.629	<.0002
BPRS item 5 (guilt feelings)	0.605	<.0004
CAPS item 23 (guilt over actions)	0.479	<.007
Mississippi item 28 ("I can't tell anyone about my military deeds")	0.564	<.001
Mississippi item 8 ("My deeds in the military deserve death")	0.463	<.01
Symptom severity measures		
MMPI-2 PTSD severity score (Keane)	0.879	<.0001
Mississippi scale total score	0.634	<.0002
MMPI-2 negative treatment indicators	0.911	<.0001

pression, Harris-Lingoes, and Supplementary Scales indicating poor social functioning and socially disabling psychological features) along with shame-guilt correlates from the DEQ, CAPS-2, BPRS, and Mississippi scales. These overall findings suggest that the MMPI-2 Content Depression Scale score may largely represent a primarily characterological measure of proneness to shame or to a shame-laden depressive syndrome in this patient sample.

Next, to improvise a broader factor that might combine the power of the best cortisol correlates from different scales, the sum of the first three depression measures listed in Table 3 (MMPI-2 Content Depression Scale, DEQ self-criticism scale, and item 15 of the Mississippi scale), was used to create a "self-critical depression factor" and placed in a standardized score matrix with cortisol values. The analysis revealed a significant inverse correlation ($r = -0.497$, $p < .005$) between this broader self-critical depression factor and urinary cortisol levels.

In regard to the Mississippi scale findings, although only two of the individual Mississippi items reflecting depressive feelings reached significance in their correlations with urinary cortisol levels, quite a few similar items reflecting depressive tendencies approached significance or showed strong trends in relation to cortisol levels. The possible usefulness of developing a "Mississippi depression factor" by operationally using items that correlated ($p < .08$) with the MMPI-2 Content Depression Scale scores was explored, and it was found that 12 Mississippi items (3, 5, 8, 12, 13, 15, 16, 18, 26, 28, 34, and 35) met this criterion. When this factor was created and entered into our matrix, it correlated significantly with urinary cortisol levels ($r = -0.375$, $p < .05$), as shown in Table 3.

When a similar procedure was carried out with the CAPS-2 scale, using intercorrelations with the MMPI-2 Content Depression Scale scores, a "CAPS-2 depression factor" was developed with 10 items (9, 10, 11, 13, 16, 17, 23, 24, 27, and 29) and was also found to correlate significantly with urinary cortisol levels ($r = -0.405$, $p < .03$).

Correlations Between Cortisol Levels and Shame and Guilt

In view of the implication above of a shame and guilt component in relation to the depression measures correlating significantly with urinary cortisol levels in this study, it is interesting that the two items in our total matrix having the strongest correlations with urinary cortisol levels are CAPS-2 item 10 (restricted range of affect; $p < .003$), discussed above as reflecting emotional numbing, and CAPS-2 item 23 (guilt over my actions; $p < .003$), a clinician assessment of guilt or shame. In pursuing these rather strong leads implicating guilt or shame, we found some additional significant correlations with other psychometric measurements believed to reflect guilt or shame in this PTSD patient sample, as summarized in Table 5. As mentioned, the CAPS-2 interviewer rating of guilt over actions correlated very significantly with urinary cortisol levels ($r = -0.527$, $p < .003$). The only other explicitly labeled guilt measure in our battery was BPRS item 28 (guilt feelings), which also correlated significantly with cortisol levels ($r = -0.418$, $p < .02$). Mississippi item 28 ("I feel there are certain things I did in the military that I can never tell anyone, because no one would ever understand") also seems to reflect feelings of guilt or shame, and it, too, correlated significantly with urinary cortisol levels ($r = -0.368$, $p < .05$).

To improvise a broader factor that might combine the power of the cortisol correlates from different scales, the sum of the three shame-guilt measures listed in Table 5 (CAPS-2 item 23, BPRS item 5, and Mississippi item 28) was used to create a "shame-guilt factor" that was placed in a standardized score matrix with cortisol values. This analysis revealed a significant inverse correlation ($r = -0.514$, $p < .004$) be-

TABLE 5. Correlations of Urinary Cortisol Levels With Psychometric Measures of Shame and Guilt in Vietnam Combat Veterans With PTSD

Shame-Quilt Subgrouping	*r*	*p*
Standard psychometric measures		
CAPS-2 item 23 (guilt over actions)	−0.527	<.003
BPRS item 5 (guilt feelings)	−0.418	<.022
Mississippi item 28 ("I can't tell anyone about my military deeds")	−0.368	<.050
CAPS-2 guilt factor (Sum of CAPS-2 items 23 and 24)	−0.415	<.023
Mississippi item 5 ("The people closest to me are afraid of me")	−0.360	<.055
Improvised psychometric factors		
Multiscale shame-guilt factor[a]	−0.514	<.004
Mississippi–CAPS-2 current life guilt factor[b]	−0.358	<.056
Mississippi–BPRS current life guilt factor[c]	−0.341	<.070
Mississippi–CAPS-2 military life guilt factor[d]	−0.256	<.180
Mississippi–BPRS military life guilt factor[e]	−0.259	<.175

[a] Sum of scores on CAPS-2 item 23, BPRS item 5, and Mississippi item 28.
[b] Sum of Mississippi items correlating closely with CAPS-2 item 23 (items 3, 5, 10, 14, 16, 22, 23, 31, 32, and 35).
[c] Sum of Mississippi items correlating closely with BPRS item 5 (items 3, 5, 23, and 31).
[d] Sum of Mississippi items correlating closely with CAPS-2 item 23 (items 2, 4, 8, 12, 28, and 29).
[e] Sum of Mississippi items correlating closely with BPRS item 5 (items 4, 8, 12, 14, 28, and 32).

tween this combined shame-guilt factor and urinary cortisol levels.

Further inspection of our correlation results also revealed some other Mississippi items besides item 28 that apparently reflect guilt feelings; these items approached but did not quite reach significance in their correlation with cortisol. Accordingly, as in the case of the depressive measures discussed above, the possibility of developing Mississippi "guilt factors" was explored, in this case operationally using the intercorrelations with, first, the CAPS-2 item 23 (guilt over my actions) score and, second, with the BPRS item 5 (guilt feelings) score.

In developing this approach, it became increasingly clear in both scales that the guilt items concerned with guilt or shame over current life actions correlated more strongly with cortisol than those having to do with past military actions. A resultant Mississippi "current life guilt factor," based on correlations between CAPS-2 item 23 (guilt over my actions) and 11 Mississippi items (3, 5, 10, 14, 15, 16, 22, 23, 31, 32, and 35), came close but did not quite reach significance in its correlation with cortisol levels ($r = -0.358$, $p < .06$). Similarly, a Mississippi current life guilt factor based on correlations between BPRS item 5 (guilt feelings) and 4 Mississippi items (3, 5, 23, and 31) was not far from significance in its correlation with cortisol levels ($r = -0.341$, $p < .07$). In contrast, the two improvised factors reflecting guilt or shame over past military actions showed correlations that were not even close to statistical significance ($p < .2$).

Table 5 also shows that the CAPS-2 guilt factor, which combines two interviewer ratings, the item 23 score (guilt over my actions) and the item 24 score (survivor guilt), does significantly correlate with cortisol levels ($r = -0.415$, $p < .02$), but this was largely due to the strong correlation with the item 23 score because the item 24 score itself was not close to significance ($p < .4$).

It should also be remembered that the significant correlations between cortisol levels and the MMPI-2 Content Depression Scale score, representing a strong shame content, as well as the DEQ self-criticism score, listed above under the self-critical depression subgrouping, are at the same time further support for the importance of this shame-guilt subgrouping in relation to cortisol levels in this PTSD sample.

Because the data seem to indicate that it is useful to distinguish military from postcombat civilian life experience in relation to guilt or shame, some of the common potential sources of guilt or shame for Vietnam combat veterans might be outlined (Table 6).

TABLE 6. Potential Sources of Shame and Guilt in Vietnam Combat-Related PTSD

Sources during military service
Participating in reprehensible or atrocious actions in the combat zone
Failure to always act bravely or to fulfill duties or expectations in combat
Being helpless, powerless, or unable to control the outcome in dire situations
Inability to protect others nearby and surviving while good friends were killed
Sources during civilian life after military service
Rejection on homecoming; public disapproval and scorn instead of thanks and honor
General inability to readjust to civilian life and live up to expected standards
Lack of control of aggressive, violent impulses, especially in harming those closest to them
Repeated failures to perform well in family or job settings
Failure to succeed in efforts to complete further education or training goals
Repeated clinical relapses with cycles of regressing after showing some improvement
Increasingly feeling the stigma of chronic disabling mental illness
Feeling a growing, hopeless sense of inadequacy, lack of control and mastery

With regard to military experience, potential sources of shame might include such factors as participation in reprehensible actions in the combat zone, especially those involving unarmed civilians; failure to always act bravely, to fulfill duties, or to prevail in combat; the perception of being helpless, powerless, and unable to control the outcome in dire situations; the inability to protect others nearby; and surviving while friends were killed.

With regard to postmilitary civilian life, perhaps even more numerous sources of shame might include such factors as rejection on homecoming; public disapproval and scorn instead of thanks and honor; general inability to readjust to civilian life and live up to expected standards; lack of control of aggressive, violent impulses, especially in harming those closest to them; repeated failures to perform well in family or job settings; failure to succeed in efforts to complete further education or training goals; repeated clinical relapses with cycles of regressing after showing some improvement; feeling the stigma of chronic disabling mental illness increasingly; and feeling a growing, hopeless sense of inadequacy, lack of control and mastery.

From the persistent or recurring everyday nature of many of the postmilitary sources of shame, it is perhaps not surprising that this phase of their life relates so strongly to the present psychoendocrine findings, which reflect their desperate resort to very costly antisocial disengagement coping strategies.

Correlations Between Urinary Cortisol and the Total Symptom Complex

When the Pearson product-moment correlation coefficient values between the three improvised psychometric factors above, the disengagement factor, the self-critical depression factor, and the shame-guilt factor, are examined, it is evident that they are so closely and positively intercorrelated as to suggest that they might well be considered to constitute a single or unified clinical or symptom complex. The disengagement factor correlates very significantly with both the self-critical depression factor ($r = 0.647$, $p < .001$) and the shame-guilt factor ($r = 0.525$, $p < .003$). Also, the self-critical depression factor correlates strongly with the shame-guilt factor ($r = 0.793$, $p < .0001$). When the sum of these three factors are combined as a triad into a single factor and placed in a standardized score matrix with cortisol values, analysis reveals the most highly significant correlation ($r = -0.610$, $p < .0003$) between any psychometric measure and urinary cortisol levels observed in this study.

Experimentation with principal component analyses of the correlational matrix also supported the conclusion that even though this multidimensional symptom complex of cortisol correlates clearly has three component subgroupings that can be distinguished clinically, the overall complex can be regarded as a single dimension, or as an integrated whole with a single-factor solution, by principal component analysis. In general, then, these findings indicate that this clinical constellation linked to cortisol levels in this PTSD patient sample is a packaged complex of closely interrelated integral components, raising the clinical question of whether they may be causally or functionally related in a psychodynamic or pathogenetic way in this disorder.

Analysis by *t* Test of High- vs. Low-Cortisol Subgroups

To further assess the validity of the correlational findings shown above, the patient sample was divided into high- and low-cortisol subgroups using a median split, and *t* test analyses were performed. Figure 2 reveals that all three of the symptom complex factors show significant differences between the two subgroups, with the low-cortisol subgroup having higher values for the disengagement factor ($t = -2.97$, $p < .006$) and the shame or shame-guilt factor ($t = -2.80$, $p < .009$) and showing a clear trend with the depression or self-critical depression factor ($t = -1.90$, $p < .07$). The overall factor representing the symptom complex as a unit, the "complex factor," also is significantly higher ($t = -3.04$, $p < .005$) in the low-cortisol subgroup. These results, obtained using a statistical procedure based on quite different assumptions and limitations than the correlational analyses presented above, contribute additional support to the validity of the relationships between this closely knit complex of clinical features and cortisol in the present PTSD patient sample.

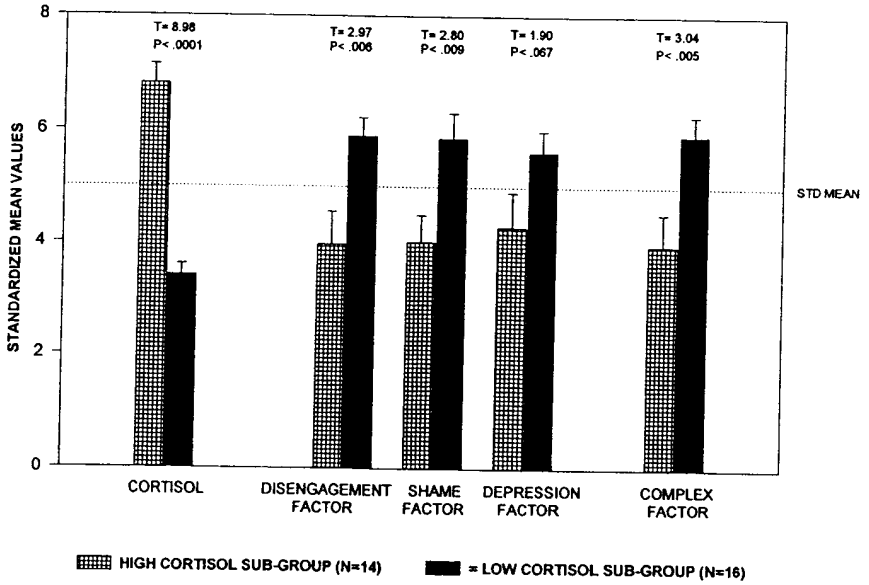

Fig. 2. Clinical psychometric differences between high- and low-cortisol subgroups in male Vietnam combat veterans with PTSD implicating the same clinical symptom complex of emotional numbing-disengagement and shame-depression that was revealed by correlation analysis. ▦ = high-cortisol subgroup ($N = 14$); ■ = low-cortisol subgroup ($N = 16$).

DISCUSSION

Relationship Between Cortisol Levels and Disengagement Coping Strategies

The primary finding in this study is the prominent relationship between emotional numbing and other disengagement coping strategies with urinary cortisol levels. The inverse nature of the correlation indicates the greater the disengagement, the lower the urinary cortisol level or vice versa. The data in the present study, based solely on associations between psychological and hormonal variables, when taken in isolation do not permit conclusions about causal relationships between these variables or about the psychogenic nature of the low cortisol levels. However, as emphasized in the Introduction, there is a compelling and substantial weight of evidence in early psychoendocrine research from which it can be reasonably concluded that the low cortisol levels observed in sustained stress typically do have a psychogenic basis. In fact, the primary finding in the present study might well have been predicted on the basis of a series of psychoendocrine studies of stress that began in 1957 with Singer's development of the construct of engagement as a broad, undifferentiated arousal process that was associated with the elevation of cortisol levels in human subjects exposed to psychosocial stress, while the use of antiarousal or disengagement defenses was associated with the lowering of cortisol levels (10). There soon followed confirmatory evidence from further stress research, including longitudinal studies of time relationships between psychosocial and cortisol changes, indicating strongly that disengagement psychological defenses, such as avoidance, withdrawal, denial, or emotional numbing, in adaptation to stressful life situations are typically associated with lowered cortisol levels and can play a dominating or overriding role in the ongoing adaptive adjustments in chronic stress (4, 9, 27).

The finding in the present study of lower urinary cortisol levels being associated with an increased inclination to use disengagement coping mechanisms also seems to provide a logical explanation for the variability of group mean urinary cortisol levels, which have been observed in various studies of patients with combat-related PTSD. It seems that the general pattern so far in the field has been to observe lower mean cortisol levels in patients when study conditions involve relatively little acutely superimposed psychosocial stress and a supportive setting in which disengagement coping mechanisms can be readily used (1, 2). On the other hand, higher mean cortisol levels are observed when greater, more potentially overwhelming psychosocial stress is superimposed, perhaps especially involving intensive research protocols or exposure treatment programs, which make it much more difficult for patients to effectively use disengagement defenses like emotional numbing, avoidance, and withdrawal (26, 28, 29). The severity of symptoms also seems to play a role in these relationships such that the pattern of longitudinal urinary cortisol change in response to the hospitalization experience seems quite different in subgroups of PTSD patients with more severe symptoms and poorer social functioning in comparison with those with better social functioning and a greater capacity to tolerate and participate in research and treatment programs (26).

Possible Functional Relationships Between Shame, Depression, and Disengagement

The related major finding of significant correlations between cortisol levels and a clinical complex that includes a shame-guilt component, a self-critical depression component, and a disengagement coping strategies component raises some important clinical questions. These clinical subgroupings do not seem on close inspection to be just randomly or separately linked to cortisol levels but rather emerge as tightly organized and coordinated integral components of a larger unitary clinical complex that, as a whole, has a prominent relationship with cortisol levels in this PTSD patient sample.

Because the findings are largely correlational, it is not possible to conclusively establish causal relationships from our present data about the nature or direction of the functional interrelationships between all of the clinical variables and cortisol levels. However, from a clinical standpoint it is reasonable to consider the way in which the disengagement coping component may be functionally related to the shame-guilt and self-critical depression clinical features that so closely intercorrelate with it as apparently integral parts of a symptom complex. It is widely recognized clinically that shame-proneness and a preoccupying fear of scrutiny and humiliation are likely to be associated with a sense of alienation, suspiciousness, and fear of others, resentment, anger, a tendency to blame and distrust others, all classic features of PTSD that in turn might be expected to lead to a strong compensatory defensive reliance on such disengagement coping mechanisms as emotional numbing, avoidance, and withdrawal from both a social and an intrapsychic standpoint.

At a very early stage in this field, Kolb (30) formulated that there may be primary and secondary relationships between some of the clinical features in chronic PTSD and that the well-known avoidance or

withdrawal behaviors may represent secondary defensive or coping strategies against the distressing primary symptom complexes, including fear, shame, and guilt. Horowitz (31) also formulated that there were two opposing sets of intrapsychic processes in response to traumatic stress, for which he used the terms "intrusion" and "denial," and he viewed denial as a secondary defensive phase triggered to counteract the primary hallmark symptom complex, the painful trauma-related memories and affects, and he recognized that this secondary defensive adaptation can become the most dominant characteristic in the chronic clinical course of traumatized individuals.

It seems that the denial process described by Horowitz (31), who referred to it alternatively as "massive emotional numbing," has since come to be more generally referred to as emotional numbing or simply numbing, and it has become widely recognized as a cardinal sign of PTSD. Keane et al. (32) conceptualizes emotional numbing symptoms as avoidance behavior serving defensively to reduce emotional distress by reducing contact with evocative external or internal stimuli. Glover (33) reports that the common terms used by patients to describe emotional numbing include being "shut down," "numb," "ice-cold," "hollow," "dead," and "empty," and there is little, if any, sense of empathic bond, relatedness, or engagement with others but rather a propensity toward alienation; feeling that they are unable to be like other people; feeling inferior to others; and seeking detachment, withdrawal, isolation, and avoidance of others and of external pressures. Glover also reviews a diverse body of inferential and indirect biological evidence suggesting the hypothetical formulation that the endogenous opiate system may play an important mediating role in emotional numbing and possibly low cortisol adaptations in PTSD.

The Self-Condemning or Shame-Laden Subtype of Depressive Syndrome in PTSD

With regard to the depressive symptoms in PTSD, the present findings lend further support to the view that although many PTSD patients may meet diagnostic criteria for major depressive disorder, there is a qualitative difference, from both a clinical and a biological standpoint, between the depressive syndrome in PTSD and that of many patients without PTSD who also meet diagnostic criteria for major depressive disorder (34). Previous studies have demonstrated that cortisol levels are lower in patients with PTSD than in patients with major depressive disorder without PTSD (1, 4, 29, 35). Furthermore, in keeping with previous observations suggesting that an overwhelming sense of self-criticism and guilt may play a central role in the depressive syndrome of many combat veterans with PTSD (34), the subtype of depression suggested by our present findings is characterized by a strong association with shame, guilt, self-criticism, self-condemnation, and seclusion, as indicated particularly by the significant correlations of urinary cortisol levels with both the Content Depression Scale scores of the MMPI-2 and the self-criticism depression scores of the DEQ, which place more weight on these shame-related clinical features than do the more widely used Hamilton or Beck scales.

In addition, the strong intercorrelations of the MMPI-2 Content Depression Scale scores with a large cluster of MMPI-2 characterological or dispositional features linked to social dysfunction raises the possibility that the shame and depressive measure scores in this disorder might also be best seen as reflecting a characterological tendency, vulnerability, or "proneness" in the PTSD patient rather than a present affective state. Under these conditions it seems reasonable to consider the possibility that simply a preoccupying fear of shame in social settings or in anticipation of social exposure may be a factor in the habitual or chronic use of disengagement coping strategies in this disorder. These findings may even raise the possibility that shame and depression in this disorder might really be best seen not as two separate features but rather as a single clinical unity, with shame-proneness representing a discriminating dimension of the depression to be viewed as a shame-laden depressive syndrome, which is qualitatively very different from other major depressive disorder syndromes.

Shame as a Neglected Clinical Feature of Combat-Related PTSD

Although guilt and shame have long been implicated as prominent clinical features in combat-related PTSD (30) and recent factor analytic studies of symptom ratings of Vietnam combat veterans have identified guilt as one of the major symptom clusters that define PTSD (36–38), neither guilt nor shame is presently included in the standard diagnostic criteria for PTSD, and their importance seems to have been underestimated in both the study and treatment of this disorder in the view of some investigators (39–42). In considering the close relationship between guilt and shame, the key concept of Lewis (43) that "shame is about the self; guilt is about things" has now been widely accepted by researchers in this field who place the focus on particular past actions as the source of guilt in contrast to having a more global focus on the entire self in shame, as might be expressed in the

difference between "if only I hadn't" (in guilt) and "if only I weren't" (in shame) (44, 45). The cognitive aspects of shame have been described as a painful awareness of oneself as defeated, deficient, exposed, a failure, inadequate, wanting, inferior, worthless, and wounded so that the very essence of the self feels wrong in the life perspective of the individual (46).

Because many veterans with PTSD seem to suffer a profound sense of regretfulness, not only about specific actions occurring in their combat experience long ago but also in their ongoing everyday life experience after their military service, and because they have developed a progressively increasing negative evaluation of themselves, it may be that the concept of shame deserves much greater attention as a central issue in this disorder. The cortisol correlations with Mississippi scale items in the present study suggest that it may indeed be these repetitive shame-inducing experiences year after year over the chronic course of the disorder that play a dominant role in perpetuating and worsening the shame-laden depression so long after the initial precipitating traumatic stress events or experiences occurred.

Horowitz (31) regarded shame and guilt as common themes in the ideational content of the intrusive phase of traumatic stress syndromes and discriminated multiple cumulative sources of guilt and shame in patients, including shame over their helplessness, powerlessness, loss of control and mastery, inability to prevent or avoid adversity, and inability to perform useful tasks reliably, and shame and guilt over their uncontrolled rage, aggressive impulses, and violent outbursts, all features that are a continuing part of the patient's life long after the original traumatic stress experience.

The recent literature on shame conveys clearly that many researchers in this field have a passionate conviction that shame is not only one of the most basic and central human affects but also a widely neglected factor or "sleeper" (43) that plays a much broader role in many forms of psychopathology than currently realized (41, 47). Tangney (48) suggests that the surprisingly little research on shame and guilt may largely be due to the problem of difficulty in measurement. Shame and guilt are deeply internal and guarded, largely hidden, affective states without clearly definable, invariable, telltale, or "codable" overt signs, and patients are typically unaware that they are experiencing shame or guilt but rather are only conscious of being painfully upset (43). Freud (49) summarized his extensive experience in this area of great interest to him by saying "patients do not feel guilty, they feel ill."

Tangney (48) points out, however, that a number of psychometrically sound instruments for the measurement of shame and guilt have been introduced recently, particularly for dispositional or characterological shame-proneness and guilt-proneness, a timely development that should facilitate closer examination of the possibility that proneness to shame and guilt are neglected clinical features that may have special importance in the study and treatment of PTSD (40, 42). The extremely refractory nature of PTSD to standard therapeutic approaches, along with the prominence of guilt, shame, and inability to receive forgiveness as clinical features, seem to have stimulated the interest of some therapists in exploring the possible usefulness of complementing psychiatric with spiritual approaches in this disorder, as suggested in a recent issue of the *Clinical Quarterly of the National Center for PTSD* (50).

Methodological and Strategic Implications of the Findings

A basic premise of the conceptual or strategic approach underlying this study is that the measurement of peripheral levels of hormones like cortisol representing specific central neuroendocrine axes can provide a much-needed objective "window into the psyche" and that each hormonal system will provide a reflection of distinctive intrapsychic mechanisms or dimensions (9, 27). The present study appears to add still further support for pursuing this view of psychoendocrine research strategy. Just as the early study of patients anticipating elective thoracic surgery led to the discovery of the new intrapsychic construct of engagement as the essential basic psychological correlate of cortisol elevation in response to psychosocial stress (10, 11), the cortisol findings in the present study implicate a very specific clinical symptom complex of disengagement and shame-laden depression as a central psychopathological feature in combat-related PTSD. This finding suggests a much greater role for the relatively hidden process of shame in the pathogenesis of this disorder than apparently has been previously suspected by many researchers in the field and provides encouragement that objective, quantitative peripheral hormonal measurements may be useful in facilitating the very difficult clinical task of assessing complex intrapsychic processes related to psychopathology, especially those more elusive clinical features without clear, telltale, overt signs or without access through self-reports of the patient.

It is also suggested that our findings have some broad methodological implications for facilitating the use of psychometric tests in clinical psychoendocrine research. First, the findings demonstrate the value of

the use of multiple psychometric instruments in the early stages of exploration of psychoendocrine relationships of a particular hormone, not as a "shotgun" strategy but to include assessment of both core diagnostic symptoms and a broader range of psychopathology, to include both state and characterological measures, to have a balance between self-reported and clinician-administered tests, and to provide confirmatory support across different tests for implicating specific clinical features as related to the hormone levels so as not to be dependent on any single item, or single-factor measure, from any single instrument for hypotheses or conclusions about psychoendocrine correlations. Second, such an approach also provides the opportunity to obtain additional information about the meaning of specific test items on the basis of intercorrelations across different scales and for improvising and evaluating new factor scores on that basis. In the present study, for example, intercorrelations between groups of Mississippi items and both the CAPS-2 guilt over my actions item and the BPRS guilt feelings item suggested the development of Mississippi guilt factors that seem to distinguish between military and post-military sources of guilt and could provide potentially useful new guilt measures that could be extracted from this widely used PTSD scale. All our findings with improvised measures, of course, will require more conclusive study to determine their validity and reliability across multiple patient samples.

CONCLUDING REMARKS

The present findings suggest that the low cortisol levels sometimes found in PTSD patients may best be conceptualized, in keeping with findings in earlier psychoendocrine research, as a reflection of the effectiveness of an intrapsychic process that may be termed disengagement (including emotional numbing, avoidance, and withdrawal coping strategies), which apparently develops as a costly secondary compensatory defensive adaptation against an overwhelmingly painful shame-laden depressive syndrome during the chronic course of this disorder. It seems to be especially important that we conceptualize cortisol levels clinically not simply as reflecting the presence or absence of arousal or intrusive symptoms in PTSD but rather as reflecting the dynamic balance between actively opposing arousal and antiarousal psychological mechanisms.

During the chronic course of PTSD, then, it seems that when the dominating forces in the balance between opposing psychological forces are the secondary, defensive, compensatory disengagement influences, this intrapsychic adaptation is reflected in lower cortisol levels. On the other hand, when, the dominating forces are the arousal or engagement influences, which may at times overwhelm defensive processes in the face of increased acute superimposed psychosocial stress, this intrapsychic adaptation is reflected in higher cortisol levels.

Cortisol levels, then, do not provide a direct "marker" for diagnosis, nor even for specific psychopathological symptoms, but basically reflect an undifferentiated intrapsychic dimension of engagement-disengagement that has an important underlying functional role in the psychopathology and pathogenesis of this diagnostic category. If this perspective is correct, it does imply some inherent limitations to exploring the use of cortisol or hypothalamic-pituitary-adrenal axis function levels as diagnostic criteria in PTSD unless systematic efforts are made to include careful evaluation of the psychosocial conditions under which the patients are being studied, the clinical state, the effectiveness of defenses at the time of study, and eventually the stages of illness in PTSD as described in the conceptual model proposed by Wang et al. (51).

Of all the hormonal systems, the pituitary-adrenal-cortical system seems to be preeminently sensitive to ongoing everyday psychosocial influences, and it is an important, immediate, practical goal to identify more systematically those milieu conditions and independent variables in clinical studies of PTSD that may have the greatest influence in changing chronic baseline urinary cortisol values with a view to minimizing such potential confounding psychosocial factors in future work in this area. Some leads along this line have emerged from the study of cortisol lability in PTSD patients at the VA National Center for PTSD as reported in a related article (26), but further work is needed to more fully identify and sort the most important clinical setting and study condition factors that may require special attention in the design of future studies so that our understanding of the meaning, interpretation, and use of cortisol measurements in the study and management of combat-related PTSD may be established on a more conclusive basis.

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